

Sibling Interaction Effects

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The effects of social interaction (*see* **Social Interaction Models**) among siblings to individual differences in behavior were first discussed by Eaves [3] and later by Carey [1] and others. In the context of behavior genetic research, social interaction effects reflect that alleles may cause variation in one or more traits of individuals carrying these alleles, but may also, through social interaction, influence the phenotypes of individuals who do not carry them [4]. Social interactions between siblings thus create an additional source of variance and generate genotype-environment covariance if the genes causing the social interaction overlap with the genes that influence the phenotype under study.

Social interaction effects between siblings can either be cooperative (imitation) or competitive (contrast), depending on whether the presence in the family of, for example, a high-scoring sibling inhibits or facilitates the behavior of the other siblings. Cooperation implies that behavior in one sibling leads to similar behavior in the other siblings. In the case of competition, the behavior in one child leads to the opposite behavior in the other child.

In the classical **twin design**, cooperation or positive interaction leads to increased twin correlations for both monozygotic (MZ) and dizygotic (DZ) twins. The relative increase is larger for DZ than for MZ correlations, and the pattern of correlations thus resembles the pattern that is seen if a trait is influenced by common environmental factors. Positive interactions have been observed for traits such as antisocial tendencies [2]. Negative sibling interaction, or competition, will result in MZ correlations, which are more than twice as high as DZ correlations, a pattern also seen in the presence of genetic **dominance**. Such a pattern of correlations has been reported in genetic studies of Attention Deficit Hyperactivity Disorder (ADHD) and related phenotypes in children (e.g., [6]). In adults, a pattern of high MZ and low DZ correlations has been observed for anger [7].

In data obtained from parental ratings of the behavior of their children, the effects of cooperation and competition may be mimicked (e.g., [8]). When parents are asked to evaluate and report upon their children's phenotype, they may compare the behavior. In this way, the behavior of one child becomes the standard against which the behavior of the other

child is rated. Parents may stress either similarities or differences between children, resulting in an apparent cooperation or competition effect.

The presence of an interaction effect, either true sibling interaction or rater bias, is indicated by differences in MZ and DZ variances. If the interaction effect is cooperative the variances of MZ and DZ twins are both inflated, and this effect is greatest on the MZ variance. The opposite is observed if the effect is competitive; MZ and DZ variances are both deflated and again this effect is greatest on the MZ variance. In addition to heterogeneity in MZ and DZ variances, the presence of interaction affects MZ and DZ correlations. Under competition MZ correlations are much larger than DZ correlations. Under cooperation MZ correlations are less than twice the DZ correlation. These patterns of correlations are not only consistent with contrast and cooperation effects, but also with genetic nonadditivity (e.g., genetic dominance) and common environmental influences, respectively. In order to distinguish between these alternatives, it thus is crucial to consider MZ and DZ variance-covariance structures in addition to MZ and DZ correlations.

Rietveld et al. [5] carried out a simulation study to determine the statistical **power** to distinguish between the two alternatives of genetic dominance and social interaction. The results showed that when both genetic dominance and contrast effects are present, genetic dominance is more likely to go undetected in the classical twin design than the interaction effect. Failure to detect the presence of genetic dominance leads to slightly biased estimates of additive genetic, unique environmental, and interaction effects (*see* **ACE Model**). Competition is more easily detected in the absence of genetic dominance. If the significance of the interaction effect is evaluated while also modeling genetic dominance, small contrast effects are likely to go undetected, resulting in a relatively large bias in estimates of the other parameters. Alternative designs, such as including pairs of unrelated siblings reared together to the classical twin study, or including the nontwin siblings of twins, increases the statistical power to detect contrast effects as well as the power to distinguish between genetic dominance and contrast effects.

Sibling interaction will go undetected in the classical twin design if the genes responsible for the interaction differ from the genes which influence the trait. In such cases, a comparison with data from singletons

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may permit further investigation. In parental ratings, the question whether an interaction effect represents true sibling interaction or rater bias also must be resolved through the collection of additional data, for example, from teachers.

References

- [1] Carey, G. (1986). Sibling imitation and contrast effects, *Behavior Genetics* **16**, 319–341.
- [2] Carey, G. (1992). Twin imitation for antisocial behavior: implications for genetic and family environment research, *Journal of Abnormal Psychology* **101**, 18–25.
- [3] Eaves, L.J. (1976). A model for sibling effects in man, *Heredity* **36**, 205–214.
- [4] Eaves, L.J., Last, K.A., Young, P.A. & Martin, N.G. (1978). Model-fitting approaches to the analysis of human behavior, *Heredity* **41**, 249–320.
- [5] Rietveld, M.J.H., Posthuma, D., Dolan, C.V. & Boomsma, D.I. (2003). ADHD: sibling interaction or dominance: an evaluation of statistical power, *Behavior Genetics* **33**, 247–255.
- [6] Rietveld, M.J.H., Hudziak, J.J., Bartels, M., van Beijsterveldt, C.E.M. & Boomsma, D.I. (2004). Heritability of attention problems in children. Longitudinal results from a study of twins age 3 to 12, *Journal of Child Psychology and Psychiatry* **45**, 577–588.
- [7] Sims, J., Boomsma, D.I., Carrol, D., Hewitt, J.K. & Turner, J.R. (1991). Genetics of type A behavior in two European countries: evidence for sibling interaction, *Behavior Genetics* **21**, 513–528.
- [8] Simonoff, E., Pickles, A., Hervas, A., Silberg, J.L., Rutter, M. & Eaves, L. (1998). Genetic influences on childhood hyperactivity: contrast effects imply parental rating bias, not sibling interaction, *Psychological Medicine* **28**, 825–837.

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